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FILE 'HOME' ENTERED AT 13:47:02 ON 16 OCT 2006

=> file reg
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                             ENTRY    SESSION
FULL ESTIMATED COST                                           0.21       0.21

FILE 'REGISTRY' ENTERED AT 13:47:31 ON 16 OCT 2006

=> e amyloid/cn
E1            1     AMYLOHEPTAOSE/CN
E2            1     AMYLOHEXAOSE/CN
E3            1 --> AMYLOID/CN
E4            1     AMYLOID (CANDIDA ALBICANS)/CN
E5            1     AMYLOID (HUMAN CLONE 72HO4 FRAGMENT)/CN
E6            1     AMYLOID (HUMAN HEREDITARY RENAL AMYLOIDOSIS-ASSOCIATED)/CN
E7            1     AMYLOID (HUMAN P-COMPONENT)/CN
E8            1     AMYLOID (MESOCRICETUS AURATUS)/CN
E9            1     AMYLOID (MONKEY) PROTEIN A/CN
E10           1     AMYLOID (MOUSE CLONE ΛMIAP-G2)/CN
E11           1     AMYLOID (MOUSE)/CN
E12           1     AMYLOID (PICHIA PINUS)/CN

=> e
E13          17     AMYLOID (SACCHAROMYCES CEREVISIAE)/CN
E14           1     AMYLOID (SYNTHETIC CLONE 10)/CN
E15           1     AMYLOID (SYNTHETIC CLONE 12)/CN
E16           1     AMYLOID (SYNTHETIC CLONE 16)/CN
E17           1     AMYLOID (SYNTHETIC CLONE 17)/CN
E18           1     AMYLOID (SYNTHETIC CLONE 19)/CN
E19           1     AMYLOID (SYNTHETIC CLONE 23)/CN
E20           1     AMYLOID (SYNTHETIC CLONE 24)/CN
E21           1     AMYLOID (SYNTHETIC CLONE 4)/CN
E22           1     AMYLOID (SYNTHETIC CLONE 66)/CN
E23           1     AMYLOID (SYNTHETIC CLONE 68)/CN
E24           1     AMYLOID (SYNTHETIC CLONE 69)/CN

=> e
E25           1     AMYLOID (SYNTHETIC CLONE 7)/CN
E26           1     AMYLOID (SYNTHETIC CLONE 71)/CN
E27           1     AMYLOID (SYNTHETIC CLONE 75)/CN
E28           1     AMYLOID (SYNTHETIC CLONE 78)/CN
E29           1     AMYLOID (SYNTHETIC CLONE 8)/CN
E30           1     AMYLOID (SYNTHETIC)/CN
E31           1     AMYLOID B (HUMAN 7-AMINO ACID N-TERMINAL FRAGMENT TRIPL
                    E REPEAT) FUSION PROTEIN WITH TETANUS TOXOID (CLOSTRIDIUM TE
                    TANI 820-844/947-967 FRAGMENT)/CN
E32           1     AMYLOID B (HUMAN 7-AMINO ACID N-TERMINAL FRAGMENT) FUSI
                    ON PROTEIN WITH 307-319-HEMAGGLUTININ (INFLUENZA VIRUS) FUSI
                    ON PROTEIN WITH ANTIGEN CS (CIRCUMSPOROZOITE) (MALARIA CIRCU
                    MSPOROZOITE) FUSION/CN
E33           1     AMYLOID B 1-42/CN
E34           1     AMYLOID B PEPTIDE(1-40) (SYNTHETIC)/CN
E35           1     AMYLOID B-PEPTIDE-BINDING ALCOHOL DEHYDROGENASE/CN
E36           1     AMYLOID B-PROTEIN PRECURSOR (HUMAN C-TERMINAL FRAGMENT)
                    /CN
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=> s e33-34 or e36
             1 "AMYLOID B 1-42"/CN
             1 "AMYLOID B PEPTIDE(1-40) (SYNTHETIC)"/CN
             1 "AMYLOID B-PROTEIN PRECURSOR (HUMAN C-TERMINAL FRAGMENT)"/C
               N
L9           3 ("AMYLOID B 1-42"/CN OR "AMYLOID B PEPTIDE(1-40) (SYNT
               HETIC)"/CN) OR "AMYLOID B-PROTEIN PRECURSOR (HUMAN C-TERMIN
              AL FRAGMENT)"/CN

=> file caplus
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                               15.16      50.25

FILE 'CAPLUS' ENTERED AT 13:53:12 ON 16 OCT 2006

=> s l9/anst
          2189 L9
       1105363 ANST/RL
L10          61 L9/ANST
                (L9 (L) ANST/RL)

=> s l10 and pd<20021106
      22742485 PD<20021106
                (PD<20021106)
L11          25 L10 AND PD<20021106

=> s l11 and (mass spec?)
         937694 MASS
          81912 MASSES
         979393 MASS
                (MASS OR MASSES)
        5351198 SPEC?
         290434 SP
           7668 SPS
         297726 SP
                (SP OR SPS)
        5564761 SPEC?
                (SPEC? OR SP)
         307183 MASS SPEC?
                (MASS(W)SPEC?)
L12          3 L11 AND (MASS SPEC?)

=> d bib abs 1-3

L12  ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2006 ACS on STN
AN   1999:777514  CAPLUS  Full-text
DN   132:148708
TI   On-line high-performance liquid chromatography/mass
     spectrometric investigation of amyloid-β peptide variants
     found in Alzheimer's disease
AU   Thompson, Andrew J.; Lim, Teck K.; Barrow, Colin J.
CS   School of Chemistry, University of Melbourne, Parkville, 3052, Australia
SO   Rapid Communications in Mass Spectrometry (1999), 13(23),
     2348-2351
     CODEN: RCMSEF; ISSN: 0951-4198
PB   John Wiley & Sons Ltd.
DT   Journal
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LA English

AB Aβ peptides are the major components of amyloid deposits in Alzheimer's disease. The presence of N-terminally truncated Aβ variants in amyloid may be a critical factor in Alzheimer's disease pathogenesis. These Aβ variants are less soluble and more amyloidogenic than full-length Aβ, making their separation, purification and identification difficult. High-performance liquid chromatog. (HPLC) at elevated temps., coupled to electrospray ionization (ES) mass spectrometry (MS), enables rapid separation and identification of N-terminally truncated Aβ variants. This methodol. provides a potential tool for exploring the importance of these Aβ variants in both the pathogenesis and diagnosis of Alzheimer's disease.

RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN

AN 1997:21587 CAPLUS Full-text

DN 126:130138

TI The profile of soluble amyloid β protein in cultured cell media. Detection and quantification of amyloid β protein and variants by immunoprecipitation-mass spectrometry

AU Wang, Rong; Sweeney, David; Gandy, Samuel E.; Sisodia, Sangram S.

CS Lab. Mass Spectrometry, Rockefeller Univ., New York, NY, 10021, USA

SO Journal of Biological Chemistry (1996), 271(50), 31894-31902
CODEN: JBCHA3; ISSN: 0021-9258

PB American Society for Biochemistry and Molecular Biology

DT Journal

LA English

AB To study the metabolism of amyloid β protein (Aβ) in Alzheimer's disease, the authors have developed a new approach for analyzing the profile of soluble Aβ and its variants. In the present method, Aβ and its variants are immunoisolated with Aβ-specific monoclonal antibodies. The identities of the Aβ variants are determined by measuring their mol. masses using matrix-assisted laser desorption ionization time-of-flight mass spectrometry. The levels of Aβ variants are determined by their relative peak intensities in mass spectrometric measurements by comparison with internal stds. of known identities and concns. The authors used this method to examine the Aβ species in conditioned media of mouse neuroblastoma cells transfected with cDNAs encoding wild type or mutant human amyloid precursor protein. In addition to human Aβ-(1-40) and Aβ-(1-42), more than 40 different human Aβ variants were identified. Endogenous murine Aβ and its variants were also identified by this approach. The present approach is a new and sensitive method to characterize the profile of soluble Aβ in conditioned media and biol. fluids. Furthermore, it allows direct measurement of each individual peptide in a peptide mixture and provides comprehensive information on the identity and concentration of Aβ and Aβ variants.

RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN

AN 1994:698351 CAPLUS Full-text

DN 121:298351

TI APP717 missense mutation affects the ratio of amyloid β protein species (Aβ1-42/43 and Aβ1-40) in familial Alzheimer's disease brain

AU Tamaoka, Akira; Odaka, Asano; Ishibashi, Yoshihiro; Usami, Mihoko; Sahara, Naruhiko; Suzuki, Nobuhiro; Nukina, Nobuyuki; Mizusawa, Hidehiro; Shoji, Shin'ichiro; et al.

CS Inst. Clinical Med., Univ. Tsukuba, Ibaraki, 305, Japan
SO Journal of Biological Chemistry (1994), 269(52), 32721-4
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB We have biochem. purified Aβ from brains of two unrelated familial Alzheimer's disease (FAD) pedigrees with the APP717 mutation (Val → Ile) and from two sporadic Alzheimer's disease (AD) brains and characterized them by means of mass spectrometry and ELISA. We observed two types of amyloid β protein (Aβ), the short-tail form (Aβ1-40) and the longtail form (Aβ1-42/43), in sporadic AD and FAD brains, and found that the ratio of the long-tail form of Aβ (Aβ1-42/43) to total Aβ was increased in FAD brains. These in vivo results were confirmed in vitro using cultured cells transfected with three kinds of APP cDNAs bearing the APP717 mutations (Val → Ile, Gly, or Phe). Taken together with the hypothesis that Aβ1-42/43 functions as "seed" that increases the kinetics of amyloid fibril formation, we conclude that the APP717 missense mutation does not create new Aβ species but promotes the increased accumulation of Aβ1-42/43 in the brain, which results in the enhancement of amyloid fibril formation from soluble Aβ. These findings provide a causal relationship between this FAD genotype and the pathol. phenotype of Aβ deposition and senile plaque formation.

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	18.78	69.03

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-2.25	-2.25

STN INTERNATIONAL LOGOFF AT 13:56:49 ON 16 OCT 2006